# THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY



SALISHAN LODGE
GLENEDEN BEACH, OREGON
1991



#### THE 53RD ANNUAL MEETING OF

THE

AMERICAN ACADEMY OF

**NEUROLOGICAL SURGERY** 

SALISHAN LODGE

**GLENEDEN BEACH, OREGON** 

SEPTEMBER 22 - 26, 1991

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## THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

September 22- 26, 1991 Salishan Lodge Gleneden Beach, Oregon

#### Sunday, September 22, 1991

1:00 p.m - 5:00 p.m. Registration and Activities Sign-up
Terrace/Long House Conference Center

1:30 p.m. - 4:30 p.m. Executive Committee Meeting Gallery

6:00 p.m. - 8:30 p.m. Welcome Reception Council House

#### Monday, September 23, 1991

7:00 a.m. - 8:00 a.m. Breakfast Business Meeting (members only)

Cedar Tree

8:00 a.m. - 5:00 p.m. Registration
Terrace/Long House Conference Center

8:00 a.m. - 1:00 p.m. Scientific Session
Terrace/Long House Conference Center

1:30 p.m. - 5:30 p.m. North Coast Tour Bus Departs Main Lobby

6:00 p.m.

Beach Party Buffet Dinner

Gleneden Beach State Park

Buses depart from Main Lobby

(casual attire; jackets recommended)

#### Tuesday, September 24, 1991

7:00 a.m. - 8:00 a.m. Breakfast Business Meeting (members only)

Cedar Tree

8:00	a.m	5:00 p.m.	Registration Terrace/Long House Conference Center
8:00	a.m. •	1:00 p.m.	Scientific Session  Long House Conference Center
9:20	a.m.		Presidential Address Nicholas T. Zervas
1:00	p.m.		Golf Tournament Sign-up Registration
1:15	p.m.		Tennis Tournament Sign-up Registration
5:30	p.m	10:00 p.m	. Dinner Salishan Lodge Dining Room Sign-up for dining times at Registration

#### Wednesday, September 25, 1991

7:00	a.m 8:00 a.m.	Breakfast Business Meeting (members only) Cedar Tree
8:00	a.m 5:00 p.m.	Registration Terrace/Long House Conference Center
8:00	a.m 1:00 p.m.	Scientific Session Long House Conference Center
1:30	p.m. 6:30 p.m.	Tour of Oregon Wine Country Bus departs from Main Lobby
7:00	p.m 8:00 p.m.	Reception Terrace Patlo/Long House Conference Center

8:00 p.m.

Annual Banquet and Dance
Long House Conference Center

Thursday, September 26, 1991

Travel Day

#### CONTINUING MEDICAL EDUCATION

The Joint Committee on Education of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons designates this continuing medical education activity for (13.5) credit hours in Category 1 toward the Continuing Education Award of the American Medical Association. The Joint Committee on Education of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

#### **GUEST ACTIVITIES**

Sunday, September 22, 1991

6:00 p.m - 8:30 p.m.

WELCOME RECEPTION
Council House

Monday, September 23, 1991

8:00 a.m. - 9:30 a.m.

**Guest Hospitality** 

Continental Breakfast

Dining Room

10:00 a.mp. - 11:30 a.m.

Salishan Lodge Chef Demonstration

Cedar Tree

1:30 p.m. - 5:30 p.m.

North Oregon Coast Tour

Bus Departs from Main Lobby

Beach Party Buffet Dinner

6:00 p.m.

Buses depart from Main Lobby

(casual attire; jackets recommended)

Tuesday, September 24, 1991

8:00 a.m. - 9:00 a.m.

**Guest Hospitality** 

Continental Breakfast

Dining Room

10:30 a.m. -12:30 p.m.

Tour to Mossy Creek Pottery and

Alder House Glass Blowing Studio

Bus departs from Main Lobby

1:00 p.m.

Golf Tournament

Sign-up at Registration

1:15 p.m. - 5:00 p.m.

Tennis Tournament

Sign-up at Registration

5:30 p.m. - 10:00 p.m.

Dinner

Salishan Lodge Dining Room Sign-up for dining times at

Registration

Wednesday.	Sentember	25	1991
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8:00 a.m. - 9:00 a.m. Guest Hospitality
Continental Breakfast
Dining Room

9:30 a.m. - 12:30 p.m. Shopping Excursion to Quality Factory
Village Outlet Shopping Mall
Bus departs from Main Lobby

1:30 p.m. - 6:30 p.m. Tour of Oregon Wine Country
Bus departs from Main Lobby

7:00 p.m. - 8:00 p.m. Reception
Terrace Patio/Long House Convention
Center

8:00 p.m. Annual Banquet and Dance
Long House Conference Center

Thursday, September 26, 1991

Travel Day

#### SCIENTIFIC PROGRAM

Monday, September 23, 1991

8:00 AM Welcome: Nicholas T. Zervas, President

#### Scientific Session 1: MODERATOR: Nicholas T. Zervas, M.D.

- 8:00 AM THE PREVENTION OF POSTLAMINECTOMY EPIDURAL FIBROSIS BY A NOVEL GLYCOSAMINOGLYCAN J.T. ROBERTSON, A.L. MERIC
- 8:20 AM BRAIN STEM CAVERNOMAS INDICATION FOR SURGERY; TECHNIQUES, INTRAOPERATIVE MONITORING R. FAHLBUSCH, C. STRAUSS, J. ROMSTOCK, W. HUK
- 8:40 AM INTRAOSSSEOUS PRESSURES IN THE LUMBAR SPINE UNDER AXIAL LOADING
  S.J. LARSON, N. YOGANANDAN, F.A. PINTAR
- 9:00 AM EXTENDED SUBTOTAL MAXILLOTOMY FOR SKULL BASE AND CRANIOCERVICAL JUNCTION ABNORMALITIES

  J.H. ROBERTSON, E.W. COCKE,JR.
- 9:20 AM MICROSURGICAL MANAGEMENT OF CRANIOPHARYNGIOMA E.L. SELJESKOG, D.Y. WEN, S.J. HAINES

#### ACADEMY AWARD PRESENTATION MODERATOR: GEORGE TINDALL

- 9:40 AM MOLECULAR STRUCTURE AND FUNCTIONAL TESTING OF HUMAN L1
  CELL ADHESION MOLECULE: AN INTERSPECIES COMPARISON
  MARY LOUISE HLAVIN
- 10:00 AM EFFECTS OF EXTERNAL PH ON IONIC CURRENTS IN SMOOTH MUSCLE
  CELLS FROM THE BASILAR ARTERY OF THE GUINEA PIG
  G. ALEXANDER WEST
- 10:15 AM COFFEE BREAK

## SCIENTIFIC Session II: SYMPOSIUM ON BRAIN TUMORS MODERATOR: ROBERTO C. HEROS

- 10:40 AM LOW GRADE SUPRATENTORIAL GLIOMAS. CURE OR PALLIATION? S. MULLAN, D. JOHNSON
- 11:00 A M IMMUNOTOXIN TREATMENT OF CENTRAL NERVOUS SYSTEM NEOPLASIA
  W.A. HALL, O. FODSTAD, A. MYKLEBUST, A. GODAL
- 11:20 AM AN UPDATE IN THE TREATMENT OF PRIMARY AND METASTATIC MALIGNANT BRAIN TUMORS USING COMBINATION CHEMOTHERAPY WITH OSMOTIC OPENING OF THE BBB E.A. NEUWELT
- 11:40 AM INTRACEREBRAL AND SYSTEMIC EFFECTS OF TUMOR NECROSIS FACTOR (TNF) SELECTED BY BRAIN TUMORS
  K.J. TRACEY
- 12:00 PM LONG TERM FOLLOWUP AFTER SURGERY FOR THIRD VENTRICULAR TUMORS
  J. AUSMAN, C. COCCIA, H. HAMIYET-CAMUSCU, G. MALIK
- 12:20 PM ANGIOGENIC MODULATION OF TUMORS IN NEUROFIBROMATOSIS R.L. MARTUZA, Y. TAKAMIYA, R.M. FRIEDLANDER, H. BREM, A.
- 12:40 PM SPECIAL LECTURE:
  INTERVENTIONAL NEURORADIOLOGY AND THE NEUROSURGEON
  L. NICK HOPKINS
  - 1:00 PM ADJOURNMENT

#### TUESDAY, SEPTEMBER 24, 1991

## SCIENTIFIC SESSION III: SYMPOSIUM ON APPLIED TECHNOLOGY MODERATOR: MARTIN WEISS

- 8:00 AM STEREOTACTIC VOP/VIM THALAMOTOMY FOR THE TREATMENT OF TREMOR
  P.J. KELLY
- 8:20 AM NEURAL NETWORKS-A UNIQUE METHOD OF DATA ANALYSIS A.R. WYLER
- 8:40 AM STEREOTACTIC TECHNIQUES IN VASCULAR NEUROSURGERY M.B. SISTI, B.M. STEIN
- 9:00 AM SPATIAL REGISTRATION OF IMAGE COORDINATE SYSTEMS AND STEREOTACTIC COORDINATE SYSTEMS USING A MACHINE VISION TECHNIQUE
  M. HEILBRUN, S. KOEHLER, P. McDONALD, W. PETERS, C. WIKER, V. SIEMENOV
- 9:20 AM PRESIDENTIAL ADDRESS: NICHOLAS T. ZERUAS INTRODUCTION: STEWART B. DUNSKER

10:10 AM COFFEE BREAK

#### SCIENTIFIC SESSION IU: MODERATOR: GEORGE OJEMANN

- 10:40 AM INTRACRANIAL HYPERTENSION AND GLIAL CELL PRODUCTION OF LEUKOTRIENE C4 INDUCED BY ENDOTOXIN
  J.B.G. GHAJAR, R.J. HARIRI, R.A.R. FRASER
- 11:00 AM MONOCLONAL ANTIBODY TO AMINOMALONIC ACID (AMA) EPITOPE REACTIVE TO ARTERIAL AND GRANULOMA FOAM CELLS: A PROBE FOR THE RADIOIMAGING OF ATHEROMAS W.M. KIRSCH, K. CHENG, S. FOWLER, T. KOCH, R. WELLERSON, S. NEHLSEN-CANNARELLA, J. VAN BUSKIRK, W. KELLN, S. GRINDE, P. WHELLAN

- 11:20 AM INTERMITTENT OR CONTINUOUS TEMPORARY VESSEL OCCLUSION: THE EFFECT OF PRE-EXISTING HYPERTENSION ON PATHOLOGICAL OUTCOME
  W.R. SELMAN, C.C. ROSENSTEIN, R.A. RATCHESON
- 11:40 AM VENTRICULAR SHUNTS FOR HYDROCEPHALUS: A CLINICAL STUDY OF THE ZERO INTRACRANIAL PRESSURE SHUNT SYSTEM E.L. FOLTZ, R. MEYER, J. BLANKS
- 12:00 PM ADENOSINE IN THE REGULATION OF NEONATAL CEREBRAL BLOOD FLOW
  T.S. PARK
- 12:20 PM SPECIAL BASIC SCIENCE LECTURE:
  MOTOR SYSTEM CONTROL MECHANISMS
  TIMOTHY J.EBNER
  - 1:00 PM ADJOURNMENT

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#### WEDNESDAY, SEPTEMBER 25, 1991

#### SCIENTIFIC SESSION U: MODERATOR: CHARLES HODGE

- 8:00 AM THE ANATOMY OF INCOMPLETELY CLIPPED ANTERIOR COMMUNICATING ARTERY ANEURYSMS W. SHUCART
- 8:20 AM STUDIES OF L-NNA AND L-ARGININE ON CEREBRAL COLLATERAL BLOOD FLOW AND ENDOTHELIUM-DEPENDENT RELAXATION M.G. MUHONEN, C.M. LOFTUS
- 8:40 AM SPONTANEOUS SEIZURES IN BABOONS, (PAPIO CYNOCEPHALUS SP):
  A NEW PRIMATE MODEL OF EPILEPSY
  S. MURK, J.L. STORY, J. LUTHER, K. KAGAN-HALLET, J. ODOM,
  E. EIDELBERG
- 9:00 AM THE MIGRATION OF FETAL ASTROCYTES AND ASTROCYTOMA CELLS THROUGH NORMAL BRAIN

  E.R. LAWS, J.J. BERNSTEIN, W. GOLDBERG

TUMORS OF THE LATERAL WALL OF THE CAVERNOUS SINUS 9:20 AM M. EL-KALLINY, H.R. vanLOVEREN, J.M. TEW, JR., J.T. KELLER

THE EARLY NEUROSURGERY OF AUSTRALIA 9:40 AM N.G.DAN

10:00 AM COFFEE BREAK

SCIENTIFIC SESSION UI: MODERATOR: STEWART B. DUNSKER

10:30 AM SPECIAL LECTURE:

STEREOTACTIC "RADIOSURGERY" AND THE NEUROSURGEON CHARLES WILSON

10:50 AM SPECIAL SYMPOSIUM:

"CONGENITAL CRANIAL BASE & SPINAL PROBLEMS

MODERATOR: STEWART B. DUNSKER

PANELISTS:

JOHN C. VANGILDER: CRANIOVERTEBRAL ANOMALIES CRANIOFACIAL ANOMALIES

JOHN A. JANE:

CHARLES J. HODGE, JR. SYRINGOMYELIA AND THE CHIARI MALFORMATION: MECHANISMS

ALBERT L. RHOTON, JR.: SYRINGOMYELIA AND THE CHIARI

MALFORMATION: TREATMENT

TETHERED CORD HAROLD J. HOFFMAN: SPINAL LIPOMAS

PAUL H. CHAPMAN:

1:00 PM ADJOURNMENT

Monday, September 23, 1991 8:00 a.m.

The Prevention of Postlaminectomy Epidural Fibrosis by a Novel Glyosaminoglycan

James T. Robertson and Albert L. Meric: Memphis, TN

The formation of epidural fibrosis following total laminectomy in New Zealand White Rabbits was significantly decreased by the intraoperative application of GL402, a synthetic glycosaminoglycan (Gliatech, Inc.,Beachwood, OH) with fibroblast inhibiting activity. Laminectomies at L2 and L4 were performed in 24 rabbits. Absorbable gelatin sponge matrix soaked with Gl402 or vehicle was applied in a blinded fashion to the operative sites. Sham operated sites (no treatment) served as controls. Animals were sacrificed after two or four weeks. The extent of epidural fibrosis was evaluated by videotaped microdissection, histological analysis and magnetic resonance imaging. Dense scar formation was evident at both time intervals in the sham and vehicle-treated sites. The sites with GL402 treatment showed practically no evidence of epidural scar formation or dural adhesions. The healing of skin and lumbodorsal fascia were not affected by treatment of the laminectomy site with GL402. These results suggested that GL402 may prove beneficial in preventing postlaminectomy pain syndromes associated with dural adhesions and epidural fibrosis.

8:20 a.m. Brain Stem Cavernomas - Indication for Surgery; Techniques, Intraoperative Monitoring R.Fahlbusch, C.Strauss, J.Romstock, W.Huk Erlangen, Germany

21 patients with brain stem cavernomas were admitted to our department: 11 underwent surgery, 10 are under observation. Diagnosis was performed by MRI with gadolinium, which revealed an additional venous malformation in 3 cases, furthermore by histological examinations.

<u>Surgery:</u> Selective radical removal was performed in the subacute phase of a recurrent bleeding and resulted in an improvement of symptoms in all but one case. This patient developed a complete VI. nerve paresis, following late surgery 3 months after a recurrent bleeding. Mesencephalic cavernomas were operated via a subtemporal or supracerebellar/velum medullare approach, pontine and pontomedullary cavernomas via a suboccipital midline approach. In the two cases with additional venous malformations the cavernoma was removed selectively.

Intraoperative neurophysiological monitoring: Monitoring of AEP and SEP, performed in all cases, covers only 20% of the brain stem pathways, including motor pathways. The surgical risk was minimized and the approach to the cavernoma improved by introducing direct stimulation of the nuclei of cranial nerves VII, XII f.i.

Observation: Since the biology and natural course of brain stem cavernomas is still under discussion, incidental cavernomas and patients, in whom hemorrhages occurred more than 2 months ago, no operation was performed (n = 10). Late surgery increases the risk of additional neurological deficits.

8:40 a.m. Intraosseous Pressures in the Lumbar Spine Under Axial Loading Sanford J. Larson, Narayan Yoganandan, Frank A Pintar Milwaukee, Wi

Recent investigations conducted in our laboratory studying the mechanics of injury of the human tumbar spine have indicted that the initiation of trauma can occur under physiologic, subfailure loads. Micro-level injury in the form of endplate fractures was demonstrated using cryomicotomy and correlated with the first onset of decrease in the stiffness of the lumbar spinal unit. The present study was undertaken to quantify and correlate the alterations in the intraosseous pressures prior to, during, and after endplate failure under single cycle loading.

Lumbar functional spinal units from fresh human cadavers were prepared by embedding the superior and inferior portions of the vertebral bodies in polymethyl methacrylate. Under fluoroscopy, 1-2 ml of radio-opaque medium was injected into the nucleus of the disc through a 22 gauge needle. A pressure gauge was introduced into the vertebral body to measure intraosseous pressure. The specimens were mounted in a custom design testing apparatus and placed under the fluroscopy unit. Axial compressive displacements were applied quasistatically. The resulting force, compression, and the vertebral pressures were recorded digitally as a function of time using a modular data acquisition system.

The specimens were loaded initially to 50% of the undeformed disc height. Following this, an acute cycle of loading was performed until endplate fracture occurred as demonstrated by passage of the contrast medium from the intervertebral disc into the vertebral body. While the overall stiffness of the structure gradually decreased, the intraosseous pressure suddenly increased indicating an accentuated load in this area. Repeated loading of the specimen simulating normal physiologic conditions also exhibited an increase in the intraosseous pressure compared to the pressures recorded from the intact specimen. These observations suggest that micro-level injury of the lumbar spine, (usually not demonstrable by conventional radiography), can be followed by raised pressure within the vertebral body. Increased pressure within bone has been implicated as a source of pain. Consequently, miniature endplate fractures may be responsible for acute low back pain in patients whose spine films appear normal.

Monday, September 23, 1991
9:00 a.m. Extended Subtotal Maxillotomy for Skull Base and Craniocervical Junction Abnormalities

Jon H.Robertson and Edwin W. Cocke, Jr. Memphis TN

The extended subtotal maxillotomy is accomplished through a degloving approach to the mid face. The maxilla is mobilized below the level of the infraorbital foramen with its blood supply preserved via the mucoperiosteum of the hard palate and contralateral tonsillar pillar. The exposure is accomplished by removal or mobilization of a part of the malar bone, coronoid process of the mandible, superior and middle turbinates, ethmoid and sphenoid sinuses, posterior nasal septum, and pterygoid plates. Extension of a retromolar incision through the anterior tonsillar pillar and lateral pharyngeal wall following mobilization of the maxilla exposes the craniocervical region from the roof of the sphenoid sinus to the fifth cervical vertebra and the skull base between each eustachian tube and carotid canal. The superior exposure allows for transposition of the temporalis muscle, bone, and skin graft to reconstruct the surgical defect if needed. The morbidity of the procedure is less than that associated with the numerous approaches described in the literature for removal of lesions involving the clivus. Cranial nerve function is preserved, and vascular injury avoided. Dysphagia and aspiration are not encountered, nor is an oroantral fistula created.

It is the purpose of this communication to describe the authors' three year experience with this direct, wide-field approach to the clivus and the upper cervical spine in the management of clivus tumors and cranlocervical junction abnormalities. A total of fourteen cases were operated upon during this period. Clivus lesions included three chordomas, four meningiomas, two low grade chondrosarcomas, one fibromyxoid tumor, one prolactin-secreting adenoma and one cystic adenocarcinoma. Two craniovertebral junction abnormalities involved the odontoid process compressing the lower brain stem. A presentation of the surgical technique in detail of this new approach to the clivus and upper cervical spine will be described. A discussion of selected cases and surgical outcome will follow.

#### 9:20 a.m. Microsurgical Management of Cranlopharyngioma Edward L. Seljeskog, Dennis Y. Wen and Stephen J. Haines Minneapolis, MN

Over the past several decades, optimal management for craniopharyngioma has and continues to remain controversial. Advances in surgical techniques, postoperative supportive care and radiologic imaging have permitted contemporary neurosurgeons to often remove these tumors with a minimum of mortality and morbidity. Although histologically a benign lesion, craniopharyngiomas have historically exhibited a major propensity for recurrence, tempting the surgeon to always consider an aggressive surgical excision has all too often resulted in major endocrine and hypothalamic dysfunction, along with significant neuropsychological alteration.

In an attempt to determine our best course for the future in the management of these lesions, we have carried out a retrospective analysis of our experience involving 34 patients, who underwent craniotomy and microsurgical excision of their craniopharyngioma from 1975 through 1989. This was a period of time when CT/MRI Imaging and microsurgical approaches were routinely used. The mean followup of these 34 cases was 6.4 years and no patient was lost to followup.

Among those 25 patients treated with surgery alone, there were ten in whom a "total resection" was felt to have been achieved. Within this group of ten patients, eight remained tumor free at followup. Two patients had recurrences, a rate of 20%.

In contrast were those 15 patients in whom residual tumor was known to have not been removed. This patient group had a recurrence rate of some 60%. In comparison were eight individuals who underwent subtotal excision, but who also had adjuvant radiation. Here there was a recurrence rate of only 12% and a significantly better recurrence free interval, compared to those patients who underwent apparent total surgical excision or subtotal excision without radiation (P<.05) and P<0.25).

Concerning endocrine and visual field defects, both of these perioperative problems were commonplace before, as well as following surgery. Our review, along with numerous other reports, clearly show that aggressive surgical excision can be both safe and effective. Still evident, however, was the rough correlation between postoperative endocrine dysfunction, and the aggressiveness of surgical excision.

While any retrospective review of this sort, cannot definitively resolve issues relating to the ideal and most effective surgical approach, it would appear from our data that when total surgical excision is not easily achieved, leaving a small residual element of tumor, coupled with postoperative radiation will not adversely effect long term patient outcome or rate of tumor recurrence.

ACADEMY AWARD Moderator: George T. Tindall
Winner: Mary Louise Hiavin
Runner up: G. Alexander West

9:40 a.m. Molecular Structure and Functional Testing of the Human L1 Cell Adhesion Molecule: An Interspecies Comparison Mary Louise Hiavin

10:00 am. Effects of External pH on Ionic Currents in Smooth Muscle Cells From the Basilar Artery of the Guines Pig G. Alexander West

10:15 a.m. Coffee Break

Scientific Session II: Symposium on Brain Tumors.
Moderator: Roberto C. Heros

10:40 a.m. Low Grade Supratentorial Gliomas. Cure or Palliation? S. Mullan and D. Johnson

There is a group of intermediate grade gliomas, mostly but not exclusively, situated in the frontal lobe, which appear to be discrete on MRI with gadolinium. They do not invade an elequent area, the internal capsule nor the basal ganglia. At surgery, by biopsy they do not infiltrate beyond their apparent bed. By a combination of MRI gadolinium (preferably 3D) for localization, mapping by evoked sensory potentials, direct electrical stimulation and local anesthesia (if necessary), for preservation of elequent cortex and by biopsy for confirmation of margins it is possible to effect an apparently total removal. Ultra sonic suction is a definite technical help. Laser and ultra sonic localization are of some but lesser value. In a small series variously diagnosed as anaplastic, intermediate, grade II, grade III and astrooligo followed for five years, there has been no sign of recurrence on the MRI gadolinium scan. It is recognized, and will be demonstrated, that such tumor free intervals do not denote a "cure". Nevertheless it is felt that this attempted "cure" is preferable to some of the alternative methods currently in use, which are biopsy or subtotal removal with irradiation, or implanted radio active isotopes.

11:00 a.m. Immunotoxin Treatment of Central Nervous System Neoplasia W.A.Hall, O.Fodstad, A. Myklebust, A. Godal Minneapolis, MN

The poor prognosis associated with primary central nervous system cancer and carcinomatous meningitis has led investigators to seek new, innovative treatment modalities. Immunotoxins are cell-type selective carrier ligands linked to extremely potent toxic proteins that have excellent in vitro and invivo efficacy against many malignant cell types. We have developed three different immunotoxins using the human growth factor, transferrin (Tfn), as the carrier ligand and an abrin variant, Pseudomonas exotoxin A (PE), and a diphtheria toxin mutant, CRM 107, as the toxin moleties. Transferrin-abrin variant and Tfn-PE inhibited protein synthesis better than Tfn-CRM 107 in LOX melanoma, SNB19 and SF295 glioma cell lines.

An animal model of leptomeningeal neoplasia was established in the nude rat using melanoma, small cell lung carcinoma (SCLC), and lymphoma cell lines. After placing an indwelling intrathecal catheter, animals were inoculated with 5x105 cells growing as a monolayer or in suspension. Lower extremity paraplegia resulted at 9.24 days for melanoma (n=20) and 25 days for SCLC (n=3). Because transferrin receptors (TR) were detected on LOX cells, one microgram of Tfn-PE was instilled intrathecally 24 hours after tumor cell inoculation. Control animals (n=10) treated with phosphate-buffered saline became paraplegic at 10.7±2.75 days, compared to treatment animals (n=10) where paralysis was seen at 15.5±4.58 days, representing a delay in onset of paraplegia of 31% (p=0.015).

The in vitro cytotoxicity of Tfn-toxin conjugates against glioma and melanoma cells supports the use of the TR as an appropriate target for these immunotoxins, until more specific cell-surface antigens are identified. The observed delay in the onset of paraplegia in treated animals with neoplastic meningitis suggests that immunotoxins could be clinically tested in phase I/II clinical trials in patients with carcinomatous meningitis.

## 11:20 s.m. An Update in the Treatment of Primary and Metastatic Malignant Brain Tumors Using Combination Chemotherapy With Osmotic Opening of the BBB

E.A.Neuwelt Portland, Oregon

Currently, 200 patients have undergone blood-brain barrier (BBB) disruption about 2,000 times. Major tumor responses have been observed in patients with malignant gliomas particularly when treated prior to radiation. The most dramatic results are in primary CNS lymphoma patients who have been included prior to radiation therapy. We have achieved a nearly 50% relapse-free survival rate (median survival 45 months) with long-term survival up to 100 months. Improved or stable cognitive function has been documented in the nonirradiated patients followed at least 1 year with serial medical psychological testing (n=8).

The choice of chemotherapeutic agents has been expanded recently in 22 patients, from methotrexate and cytoxan to include etoposide and carboplatinum. Germ cell tumors have been particularly responsive to these latter drugs and objective responses also have been seen with lymphoma and breast cancer. We are interested in treating patients with metastatic breast cancer and lung cancer with carboplatinum and etoposide in view of the sensitivity of these carcinomas to these agents. The addition of carboplatinum and etoposide, as well as the recent introduction of bone marrow growth factors (i.e., G-CSF), has significantly expanded our ability to deliver effective solid tumor agents in both primary CNS disease and metastatic disease prior to radiotherapy with less myelosuppression and with maintenance of cognitive function.

11:40 a.m. Intracerebral and Systemic Effects of Tumor Necrosis Factor (TNF) Secreted by Brain Tumors K.J.Tracey Nre York NY

TNF, a cytokine produced by astrocytes, hypothalamic neurons, and inflammatory cells, is involved in the host response to meningitis, AIDS, head injury, edema formation, and multiple sclerosis. Because it is also cytotoxic against certain tumors, there is some interest in its role as a potential anti-neoplastic agent. Very little is known however, about the local or systemic effects of TNF produced in the region of a brain tumor. We have studied these effects using a genetically engineered cell line (CHO-TNF) that secretes h-TNF. When stereotactically injected into the brains of nude mice, these cells form TNF-producing brain tumors; control mice received genetically similar cells (CHO-NEO) that differed only by the absence of the h-TNF gene. RESULTS: h-TNF produced by CHO-TNF brain tumors crossed the blood-brain barrier, resulting in marked elevations of serum h-TNF (0.66  $\pm$  0.22 ng/ml); no h-TNF was detectable in controls. The local production of h-TNF in brain induced several striking histological changes, most notably peritumoral neovascularity, dilated blood vessels with focal thrombosis in adjacent microvasculature, and gliosis. These changes were not observed in controls with similar-sized CHO-NEO tumors, which grew at similar rates. Mice bearing TNFsecreting tumors succumbed within days however, because they developed profound anorexia and weight loss. Interestingly, an entirely different metabolic response developed when the TNFsecreting tumor was implanted in the hindlimb: although serum TNF levels were similarly elevated, anorexia and weight loss did not develop for eight weeks. CONCLUSION: TNF secretion in the region of a brain tumor causes prominent neovascularity and local inflammatory responses. Moreover, TNF-induced alterations of CNS neuroendocrine outflow caused profound derangements of metabolic homeostasis independent of circulating peripheral TNF. These studies describe a useful model for investigating the biology of TNF in intracerebral tumor growth. It is hoped that additional studies may advance our understanding of the role of cytokines in the pathogenesis of brain tumors.

12:00 p.m. Long Term Followup After Surgery for Third Ventricular Tumors J.Ausman, C.Coccia, H. Hamiyet-Camuscu, G. Malik Detroit Mi

Many series of open microsurgical resection of third ventricular lesions have been published. In this series we have had the opportunity to review treatment of tumors from all regions of the third ventricle at long-term follow-up with a special emphasis on the patient's ability to return to and maintain over several years his premorbid level of function. From 1979 to 1990, 49 patients underwent surgical procedures for third ventricular region neoplasms excluding craniopharyngioma and pituitary adenomas. For analysis the lesions were divided into colloid cysts (16), anterior and mid-third ventricular tumors (13), and pineal region tumors

(20). For all groups long-term survival was primarily dependent on the natural history of the histopathology. At follow-up (clinic interview and exam or phone interview if not available for clinic exam) function was assessed as excellent (back to work and/or pre-morbid level of function), good (able to work, some at original job but have some deficit which impairs performances), fair (significant deficits but able to care for self, may be able to hold mental icb), and poor/dead.

In all groups morbidity in long-term survivors were primarily related to cognitive and memory deficits related to surgery. At a mean follow-up of 4.7 years, 23% of colloid cyst patients were so impaired that they were no longer capable of reasonable work and another 15% had deficits preventing them from attaining their pre-morbid level of function. Of the 11 of 13 patients with anterior or mid-third ventricular lesions assessed at a mean of 7.6 years, 27% had excellent function, 27% good, 18% fair, and 27% were dear or poor. At a mean follow-up of 5.4 years, over half of the pineal tumor patients ere functioning well but the significance of this is small since the histopatholgy is so variable in this region and survival is very dependent on patholgy. More importantly, 10 of 11 long-term survivors are functioning well (5-excellent, 5-good) at follow-up.

Open resection of third ventricular lesions is known to be technically possible. The standard open and perhaps even the limited approach resection of colloid cysts can leave patients with very subtle but profoundly disabiling deficits. Because of the varied pathology and frequency of relative radioresistance of pineal region tumors coupled with 90% of long-term survivors functioning well, open resection remains the procedure of choice for initial treatment of these lesions. Anterior and mid-third ventricular tumors can also be treated acceptably with standard open resection; however, new image guided stereotaxic resection may allow for more extensive resection with a lower morbidity.

12:20 p.m. Angiogenic Modulation of Tumors in Neurofibromatosis R.L.Martuza, Y.Takamiya, R.M.Friedlander, H.Brem,A.Malik Boston MA

There has been neither a useful animal model for neurofibromatosis (NF) nor a beneficial nonsurgical therapy for NF and NF-associated tumors. We have used a human tumor/nude mouse xenograft model to study growth and neovascularization of benign and malignant human schwann cell tumors from both NF1 and NF2 as well as tumors of similar histologic type from non-NF patients. In prior reports, we demonstrated that tumor "take" and growth for schwannomas (acoustic neuromas) correlated with the extent of induction of neovascularization. We also demonstrated that inhibition of neovascularization with heparin and cortisone also inhibited growth of a human malignant schwann cell tumor (neurofibrosarcoma). However, this regimen

is not applicable to human use. Using human acoustic neuromas and neurofibromas, we now present data demonstrating that the anglogenic modulator, AGM-1470, will inhibit neovascularization in this model. Our initial studies suggest that AGM-1470 is effective both in neurofibromas from NF1 and acoustic neuromas from NF2 patients as well as from histologically similar tumors not occurring in NF. Further, in one human neurofibrosarcoma from a patient with NF1, we have demonstrated growth inhibition by AGM-1470. Administration of AGM-1470 is well-tolerated and is compatible with human use. Angiogenic tumor growth modulation provides a novel means of therapy for both benign and malignant tumors of the central and peripheral nervous system in people with NF1 or NF2 and for histologically similar tumors in the general population.

12:40 a.m. SPECIAL LECTURE

Interventional Neuroradiology and the Neurosurgeon

L.Nick Hopkins Buffalo NY

1:00 p.m. Adjournment

Tuesday, September 24, 1991

SCIENTIFIC SESSION III

SYMPOSIUMON APPLIED TECHNOLOGY MODERATOR: MARTIN WEISS

8:00 a.m. Stereotactic VOP/VIM Thalamotomy for the Treatment of Tremor in the Post-L-Dopa Era P.J. Kelly Rochester MN

Following the general availability of L-dopa in 1968, ventralis lateralis (VL) thalamotomy has been only rarely offered to patients with medically refractory tremor. However, new stereotactic instrumentation and improved stereotactic localization methods which employ computer-based imaging, neurophysiologic techniques and intraoperative speech monitoring render these procedures safer now than in the past.

Seventy three patients underwent stereotactic ventralis lateralis thalamotomy (24 right, 44 left, 5 bilateral) at Mayo Clinic between September 1984 and March 1991 for the treatment of medically refractory movement disorders, including 42 patients with Parkinson's disease, 8 patients with essential tremor, 17 patients with intention tremor, and 6 patients with complex dystonias. Long term postoperative follow-up examinations (mean follow-up 28 months) in each of these groups revealed significant improvement (complete ablation) in 39 (36) of 42

Parkinson's patients, 8 (4) of 8 essential tremor patients, 15 (13) of the 17 patients with Intention tremor and in 4 (3) of the 6 patients with dystonia. There was no mortality. Morbidity in this series includes 2 patients with contralateral limb dyspraxia (mild/moderate), and 4 patients with mild to moderate hypokinetic dysarthria following left thalamotomy. Modern VL thalamotomy provides an excellent surgical alternative to patients with parkinsonian tremor, familial or essential tremor, and intention tremor.

## 8:20 a.m. Neural Networks--A Unique Method of Data Analysis A.R.Wyler Memphis TN

A neural network is a form of computer simulated artificial intelligence that looks for complex relationships between numerous variables. Once given a set of variables, the network begins to learn relationships between them and can be trained to generalize from them. This approach is especially good for pattern recognition and completing incomplete data sets. Neural networks have gained increasing interest from a variety of scientific disciplines and have been developed to solve such complex problems as predicting protein structure from amino acid sequences. We have begun developing neural network to help select candidates for epilepsy surgery. Preliminary results show that neural networking can predict with a high degree of accuracy which patients will or will not do well with resective surgery. The theoretical background to neural networks and how they may be applied to other clinical problems will be discussed.

### 8:40 a.m. Stereotactic Techniques In Vascular Neurosurgery M.B.Sisti and Bennett M. Stein New York, NY

Improvements in stereotactic techniques have expanded the indication of these methods in the management of arteriovenous malformations (AVMs) and cavernous malformations (CMFs). Primarily, the precision of stereotactic localization can be utilized for either the microsurgical resection of small or inaccessible vascular lesions or for use with radiosurgical devices for similar purposes. Although much attention has recently been devoted to the stereotactic radiosurgery of AVMs and CMFs, less attention has been directed to the stereotactically guided resection of these lesions. Both radiosurgery and stereotactically guided microneurosurgery have been available at our institution for the last 2 years. During this period 25 CMFs and 10 AVMs were resected by stereotactically guided craniotomy and 1 CMF and 8 AVMs were treated with a linac radiosurgery unit.

The primary indications for the use of stereotactic localization in this group of 44 patients were lesion size and location. There is a substantial overlap in the use of either radiosurgery or

stereotactically guided craniotomy in the treatment of AVMs or CMFs less than 2.5 cm in diameter. Stereotactically guided craniotomy for the complete resection of these lesions was used whenever possible and radiosurgery was reserved for lesions which were larger, more irregular, and those lesions that involved the internal capsule, thalamus, or brain stem. In general, patients treated with radiosurgery did not present with intracerebral hemorrhage as their primary presenting symptom, whereas the group treated with craniotomy presented with symptomatic hemorrhages from their vascular malformations.

The operative results, indications and follow-up of this group of patients will be presented. The indications for stereotactically guided craniotomy vs. radiosurgery will be presented with regards to the management of small AVMs and CMFs.

9:00 a.m. Spatial Registration of Image Coordinate Systems and Stereotactic Coordinate Systems Using a Machine Vision Technique

M.Heilbrun, S.Koehler, P.McDonald, W.Peters, C.Wiker, V. Slemonov Salt Lake City, UT

CT and nondistorted MRI image data sets can be gathered as a volume array that can be defined as a Cartesian image coordinate system. Through computer graphic techniques external fiduciary marks such as natural facial structures or markers noninvasively fixed to scalp can be assigned a coordinate in the image coordinate system which can be related to the position of intracranial structure. A machine vision technique used in the industrial workplace as a three dimensional digitizer can be used to define a stereotactic coordinate system. This technique uses a perspective transformation to determine the three dimensional position of objects seen on digitized video images. Such objects can be the identical external fiduciary marks defined in the image coordinate system. Using computers for spatial registration of Image coordinate systems with stereotactic coordinate systems has been termed frameless stereotaxy.

We have described initial accuracy tests of stereotactic localization with video cameras tested with a standard BRW simulator and a phantom using a specially constructed video localizer similar to the Gybels-Suetens and Siddon stereotactic angiographic localizers. Localization accuracy was within two millimeters.

Initial experience with this technique will be described. With PC and Macintosh computers these techniques can be used for monitoring the position of orientation of instruments within the cranial vault without the constraint of a stereotactic arc.

With sufficient computer speed these techniques can be used for continuous registration on images of the position of a surgical instrument.

9:20 a.m. PRESIDENTIAL ADDRESS: NICHOLAS T. ZERVAS

10:10 a.m. Coffee Break

SCIENTIFIC SESSION IV: MODERATOR: GEORGE OJEMANN, M.D.

10:40 a.m. Intracranial Hypertension and Gliai Cell Production of Leukotriene C4 Induced by Endotoxin

J.B.G.Ghajar, R.J.Hariri, R.A.R.Fraser New York, NY

Marked deterioration of neurologic function accompanies organ dysfunction in systemic infection. Although previous hypotheses have suggested that either cerebral hypoperfusion. anoxia, or progressive brain edema may be causative, the pathogenesis remains unknown, Septic patients with stable or supported hemodynamics and adequate oxygenation may manifest confusion, stupor, or coma. Many mediators of inflammation have been implicated in derangements of host response which precede organ dysfunction in systemic sepsis. Leukotrienes (LTs) have been identified as mediators of the cerebrovascular dysfunction in models of central nervous system malignancy and traumatic brain injury, which may be characterized by alternations in blood brain barrier function leading to increased brain free water content. We previously demonstrated that cultured human glial cells generate LTC4 de novo in response to mechanical injury and calcium ionophore A23187. The acid (AA) from membrane phospholipid pools. Endotoxin (LPS) is a specific phospholipase A2 stimulant, the enzyme responsible for release of AA from membrane phospholipids. These studies were performed to test the hypothesis that neurologic dysfunction associated with sensis is mediated by similar alterations in glial cell metabolism stimulated by LPS exposure resulting in the generation of these lipoxygenase products in vitro and whether endotoxemia in miniature swine was associated with intracranial hypertension associated with the appearance of these elcosanoids in cerebrospinal fluid. Confluent cultures of human glial cells obtained from normal brain surgical specimens were employed in this study. Baseline supernatants were collected and cultures were exposed to varying concentrations of endotoxin in serum free media. Supernatants were collected at 5, 15, 30 and 60 minutes following exposure and evaluated using a competitive displacement enzyme immunoassay. LPS stimulated glial cell LTC4 production at doses>100pg/ml supernatant. Sexually mature miniature vucatan swine were surgically prepared as previously described (Hariri, et al, Surg. Forum, Vol 38, 1989). Animals were then exposed to endotoxin (LPS W E. Coli 0127:B8) 0.1 mg/kg t.V. with hemodynamic and Intracranial pressure monitoring. Lactated Ringer's solution was infused to maintain a mean arterial blood pressure of 55 mm Hg. Intracranial pressure rose dramatically within 3 hours of endotoxemia despite significant hypotension. In addition, CSF levels of LTC4

#### September 24, 1991

were significantly elevated above baseline 4 hours after endotoxin exposure compared to baseline values. These data indicated that: 1-concentrations of LPS greater than 100 pg/ml can stimulate the production of LTC4 from glial cells in similar amounts to that seen following mechanical injury and ionophore exposure. 2-endotoxemia results in intracranial hypertension associated with the appearance of LTC4 in CSF. This strongly suggests a role for LPS induced leukotriene production by human glial in the pathophysiology of cerebrovascular dysfunction and neurologic derangements associated with seosis.

11:00 a.m. Monoclonal Antibody to Aminomalonic Acid (AMA) Epitope Reactive to Arterial and Granuloma Foam Cells: A Probe for the Radiolmaging of Atheromas

W.M.Kirsch, K.Cheng, S. Fowler, T.Koch, R. Wellerson, S.Nehlsen-Cannarella, J.VanBuskirk, W. Kelln, S.Grinde, P.Wheelan. Albuquerque, NM

Aminomalonic acid (AMA) is an acid labile amino recently identified in protein hydrolysates of calcified tissues including atherosclerotic plaque. We have prepared hybridomas producing monoclonal antibodies (MoAbs) to the AMA epitope (anti-Ama). An Ama-containing peptide (-NH-CS-NH-C<sub>8</sub>-NH-CS-NH-(CH<sub>2</sub>)<sub>5</sub>-CO-NH-CH(CO<sub>2</sub>H)-CO-NHEt) has been attached to lysine residues of bovine serum albumin and keyhole limpet hemocyanin at high epitope densities (molar ratios of Ama to protein: 40 - 50). A panel of monoclonal antibodies has been produced that exhibit high affinity to peptide-bound Ama (on the order 10-9 M) with some cross reactivity to synthetic peptide bound aspartyl or glutanyl residues. Immunocytochemical studies using anti-Ama MoAb reveal striking reactivity with arterial foam cells in four species (human, subhuman primate, rabbit, and rat), in addition to experimentally induced foam cell granulomas.

I-131 anti-Ama labelled MoAb (IgG<sub>2</sub>) has been administered to both homozygous EHL-WW atherosclerotic rabbits and normal NZW rabbits. Sequential dynamic studies with the Picker Gamma Camera provided imaging until the animals were sacrificed and the major organs harvested. Though higher specific binding to the atheromatous arteries was detected, background imaging reduced the target/non-target ratio to approximately 1.2 to 1.3. This immunopharmacokinetic problem is being currently studied by utilizing both smaller immunoreactive fragments and preloading with cold MoAb. This study attempts to image the atheromatous lesion at early stages of its evolution when reversibility or halting of the disease may be accomplished.

11:20 a.m. Intermittent or Continuous Temporary Vessel Occlusion? The Effect of Preexisting Hypertension on Pathological Outcome W.R.Selman, C.Rosenstein, R.A.Ratcheson Cleveland, Chio

#### INTRODUCTION:

Temporary occlusion has become a standard procedure in the treatment of difficult aneurysms. The optimal method of temporary vessel occlusion has not been determined since the tolerance of the brain to focal ischemia is not well defined, the role of reperfusion injury may be significant, and differences in the baseline cerebrovascular status may influence the response to temporary occlusion. In order to elucidate the role of these factors, we developed a small animal model of temporary, reversible, focal ischemia.

#### METHODS:

Four groups of male, wistar rats, underwent middle cerebral artery occlusion. Two strains of rats were used; spontaneously hypertensive (SH) and normotensive (NT). Two paradigms of vessel occlusion were used; one episode of 60 minutes duration or 3 episodes of 20 minutes of occlusion with 10 minutes of reflow between each episode. Cerebral blood flow was monitored with a laser doppler flow probe over the affected hemisphere. Pathological analysis for volume of infarction was determined 72 hours after the ischemic insult.

#### RESULTS:

Blood flow was decreased in all animals in the affected territory, and reperfusion was confirmed in all multiple occlusion animals. The volumes of infarction were as follows: SH-multiple (n=6) 220±11 mm³; SH-single (n=5) 195±26 mm³; NT-multiple (n=5) 98±19 mm³ NT-single (n=5) 164±24 mm³. Five of six multiple SH animals demonstrated severe hemorrhagic infarction in the affected hemisphere.

#### CONCLUSIONS:

Intermittent, temporary occlusion is associated with a smaller volume of infarction in normotensive animals. In hypertensive animals, although the volumes of infarction were similar, intermittent temporary occlusion may actually be more deleterious in light of the hemorrhagic nature of the infarction.

11:40 a.m. Ventricular Shunts for Hydrocephalus: A Clinical Study of the Zero Intracranial Pressure Shunt System E.L.Foltz, R.Meyer, J.Blanks Orange CA

Overdrainage of CSF by ventricular shunts is increasingly recognized, though the incidence is still somewhat imprecise. Subdural hematoma, acquired cranicstenosis, sitt-ventricle syndrome and low ICP syndrome may result. The overall incidence is 10-15% occurring at 6 1/2 to 7 years after initial shunt. Acquired disability can be severe.

Our report in 1987 identified the low ICP syndrome in 14 patients. A Fio-Control valve was initially effective in this group, but the low ICP syndrome returned in 9 of the 14 patients within two years, apparently due to the valve still functioning on a CSF pressure gradient.

Therefore, ICP in normal patients as compared to those with ventricular shunts for hydrocephalus was reexamined. Emphasis appeared concerning the difference in ICP in supine and upright body positions. Gravity induced CSF shunt flow clearly played a major role in hydrocephalus shunt overdrainage, resulting in an unusual degree of negative ICP.

The concept of a "zero intracranial pressure level" which could be varied and controlled by use of a shunt which included a device already on the market (Siphon Control Device) resulted in the Zero Pressure Shunt System. This system should control ICP by closing shunt function at a selected negative ICP in the <u>body upright position</u> (determined ONLY by device position on the skull relative to gravity). The opening pressure of the shunt functions in the <u>supine body position</u> controlled only by a standard reservoir - low pressure Flo-Control valve. This system has a selected opening pressure (upright body) and a closing pressure (supine body; Siphon Control Device).

We report the use of this system in 58 patients with obstructive hydrocephalus. This includes indications for use, accuracy of estimated and achieved post-operative ICP, effect on ventricle size (CT), complications and clinical effectiveness over the past four years. Comparisons are made with 27 "control patients" where possible.

## 12:00 p.m. Adenosine in the Regulation of Neonatal Cerebral Blood Flow T.S.Park St. Louis MO

Our laboratory has demonstrated evidence that adenosine, a purine nucleoside, is involved in mediation of vasodilatory adjustments during hypotension, hypoxia and seizures in the neonate. In newborn piglets subjected to hemorrhagic hypotension, we measured interstitial fluid adenosine concentrations of the cerebral cortex using the microdialysis technique and pial anteriolar diameters using the cranial window technique. Hemorrhagic hypotension caused progressive increases in cortical interstitial adenosine concentrations in parallel with progressive dilation of the pial arterioles. Furthermore, the pial arteriolar dilation in

response to hypotension was enhanced by topical application of dipyridamole, an adenosine transport inhibitor. Detailed experimental data supporting adenosine as a metabolic mediator for regnatal cerebral blood flow will be presented.

## 12:20 p.m. Intraoperative Angiography as an Adjunct to Vascular Neurosurgery Mechanisms

D.L. Barrow Atlanta GA

Intraoperative angiography is useful in verifying the results of neurovascular operations during the procedure and prior to wound closure. In the past, the popularity of intraoperative angiography has been hindered by technical limitations such as bulky, expensive equipment, inefficient film cassettes, lengthy development time, the need for large volumes of contrast, poor resolution and obscuration of angiographic detail by radiopaque surgical implements.

We report our technique and experience with 141 intraoperative angiographic procedures performed with a portable digital subtraction unit that has eliminated many of the earlier detractors to the routine use of the technique. Of these 141 studies, intraoperative angiography provided information that altered the operative procedure on 25 occasions, thus reducing the need for reoperations and potentially reducing operative complications. In seventy cases, postoperative angiography was performed and compared to intraoperative studies.

We have found intraoperative angiography to be valuable in documenting the complete obliteration of intracranial aneurysms and both intracranial and spinal AVM's, assuring the patency of normal vessels in the vicinity of vascular anomalies, verifying the patency of endarterectomies and bypass grafts, for intraoperative localization of small vascular tesions that are not readily apparent on the surface of the brain or spinal cord, and for visualization during intraoperative endovascular procedures.

The quality of images provided by digital subtraction angiography makes the technique a useful adjunct to neurovascular surgery.

12:40 p.m. <u>Special Basic Science Lecture:</u> Motor System Control Mechanisms T.J. Ebner Minneapolis MN

1:00 p.m. Adjournment

#### WEDNESDAY, SEPTEMBER 25, 1991

SCIENTIFIC SESSION V MODERATOR: CHARLES HODGE

8:00 s.m. The Anstomy of Incompletely Clipped Anterior Communicating Artery Anaurysms
W.Shucart Boston, MA

In a series of 132 patients with anterior communicating artery aneurysms the author has had an Incidence of Incompletely clipped aneurysms of approximately 6%. Seven of those eight cases had similar anatomic set ups which were related to the incomplete clipping. This situation was one in which the aneurysm pointed superiorly and posteriorly. This anatomy makes it easy for the surgeon to only visualize part of the dome of the aneurysm while thinking he is visualizing all the dome. Case illustrations and ways to avoid the problem will be presented,

## 8:20 a.m. Studies of L-NNA and L-arginine on Cerebral Collateral Blood Flow and Endothelium-Dependent Relaxation M.G.Muhonen, C.M.Loftus Iowa City Iowa

Nitric oxide (NO) is postulated to act as an endothelium-derived relaxing factor (EDRF). This study tested the hypothesis that inhibition of NO formation with NG-nitro-L-arginine (L-NNA, which blocks synthesis of NO) during hypothesion would decrease collateral cerebral blood flow, and that this effect would be reversed by administration of L-arginine (a precursor in NO synthesis). In 25 procedure of the process of the procedure of the process of the procedure of the procedure

and that this effect would be reversed by administration of L-arginine (a precursor in NO synthesis). In 25 anesthetized dogs, a branch of the middle cerebral artery was cannulated, and the collateral dependent tissues (CDZ) were identified with a shadow flow technique. Regional cerebral blood flow (rCBF) was determined with radiolabeled microspheres. In five animals with phlebotomy induced hypotension (MAP 50 mmHg), CDZ rCBF decreased from 70±22 ml min-1 100g -1. (mean±SE) to 55±18 and 46±17 at 20 and 40 minutes after injection of L-NNA 5 mg/kg (p<0.05). Vessel diameter decreased insignificantly and microvascular pressure did not change. When both L-NNA and L-arginine (60 mg/kg) were infused in five separate hypotensive dogs rCBF increased from 63±20 to 83±18 and 91±17 at 20 and 40 minutes (P<0.05 for both), with an insignificant increase in vessel diameter.

Fifteen animals were then studies under normotensive conditions. Although vessel diameter decreased 13% after L-NNA infusion, and increased 18% after L-arginine infusion, there was no significant change in CDZ rCBF.

There were two major findings from these studie. First, inhibition of NO significantly decreased rCBF under demand (hypotensive) but not normal conditions. Second, L-arginine produced

significant increases in rCBF during hypotension. The hypothesis was proved, and these finding support the role of L-arginine derived nitric oxide in mediating vasorelaxation under demand conditions, in this case hypotension.

8:40 a.m. Spontaneous Selzures in Baboons (Papio Cynocephalus SP): A New Primate Model of Epilepsy

S.Murk, J.L.Story, J.Luther, K.Kagan-Hallet, J.Odom, E. Eidelberg San Antonio, Texas

A unique group of babcons (Papio cynocephalus sp.) in a large colony of the Southwest Foundation for Biomedical Research, display typical grand mal selzures without any obvious provocation. Most of the animals studied so far seem to also have frequent episodes suggestive of Several animals have been monitored with scalp and/or complex partial seizures. stereotactically implanted deep brain electrodes, with simultaneous video and EEG recordings to correlate the electro-physiologic substrate with the observed clinical seizure activity. Anaylis of this data suggests that the origin of these spontaneouly occuring seizure events lie in the mesial temporal lobe structures. Neuropathologic evaluation with serial section studies of the brains of these animals, including two who died in status epilepticus, showed no obvious pathology with usual light microscopic studies including Nissl and Golgi strains. GABA receptor binding and enhancement of specific flunitrazepam binding by GABA have been studied in these animals in an attempt to determine the possible involvement of these receptor systems in the seizures. Preliminary studies suggest lower levels of GABA binding in the cerebral cortex of epileptic baboons relative to controls, and decreased coupling between GABA and benzodiazepine receptor sites in cortex and hippocampus.

C-fos levels have been obtained from several regions of epileptic and control baboon brain as well and results indicate a several fold increase in the hippocampus of seizure animals over controls. Analysis of the breeding colony database has yielded preliminary evidence that the seizure phenomena may be genetically determined.

Data from on-going studies is presented, with examples of scalp and depth electrode EEG, light microscopic pathologic analysis of serial brain sections as well as receptor systems studies and C-fos measurements from multiple cerebral locations. Pedigree data tracing the appearance of seizure affected animals through several generations of the breeding colony at Southwest Foundation for Biomedical Research is also presented.

9:00 s.m. The Migration of Fetal Astrocytes and Astrocytoma Cells through Normal Brain

E.R.Laws, J.J.Bernstein, W.Goldberg Washington DC

We performed transplantation studies of labeled fetal rat astrocytes in normal rat brain which revealed impressive migration of transplanted graft-derived cells moving in stereotypical pathways over basal lamina and along white matter tracts. Similar studies were performed using C6 astrocytoma cells, and their migratory ability was even more impressive, with tumor cells migrating long distances by creating tunnels and tubes through matrix tissues of the brain, including basement membrane. We now report studies of C6 cells migrating through hydrated collagen I get and through artificial basement membrane, with further insights as to the mechanisms involved and the tumor cell-specific nature of such migratory activity. It is of great interest that artificial basement membrane is an effective barrier for fetal rat astrocytes but not for astrocytoma cells.

The studies of tumor cells migrating through brain matrix may be a model for the phenomenon of multifocal glioma and also for recurrence of malignant glioma after effective local therapy. In the experimental situation, individual cells migrate through matrix tissues, and secondarily form masses by coalescence of adjacent micropockets of tumor colls. The collagenases, proteases, cell adhesion molecules and other basic mechanisms involved are currently under study.

9:20 s.m. Tumors of the Lateral Wall of the Cavernous Sinus M.El-Kalliny, H.R.vanLovern, J.M.Tew,Jr., J.T.Keller Cincinnati, Ohio

The lateral dural wall of the cavernous sinus (CS) is composed of two layers: the outer dural layer (dura propria) and the inner membranous layer. The membranous layer is formed by the sheathes of cranial nerves III, IV,  $V_1$  and occasionally  $V_2$  and a reticular membrane extending between these nerve sheaths. These two layers are loosely attached and easily separable. Tumors arising from the contents of the lateral dural wall are located between these two layers and are classified as interdural. They are in essence extradural/extracavernous. The inner membranous layer separates these tumors from the venous channels of the CS.

Preoperative recognition of tumors in this location is critical to selection of an appropriate microsurgical approach. The MRI characteristics of interdural tumors include an oval shaped, smooth bordered mass with medial displacement but not encasement of the cavernous internal carotid artery. Tumors in this location can be safely resected without entering the CS proper by techniques which permit reflection of the dural propria of the lateral wall.

During the last four years we have identified and treated five patients with interdural cavernous sinus tumors: two trigeminal neurinomas arising from  $V_1$ , two epidermoid tumors, and one malignant melanoma presumed primary. The authors will discuss the pathoanatomic characteristics, the clinical/radiologic findings, and the microsurgical approach of this unique group of tumors. Comparison of the results of treatment of interdural versus other CS tumors documents a more favorable prognosis for tumor resection and cranial nerve preservation for those which are interdural.

9:40 a.m. The Early Neurosurgery of Australia N.G. Dan Sydney, Australia

10:00 a.m. Coffee Break

SCIENTIFIC SESSION VI: MODERATOR: STEWART B. DUNSKER

10:30 a.m. SPECIAL LECTURE:

Stereotactic "Radiosurgery" and the Neurosurgeon Charles Wilson San Francisco, CA

10:50 a.m. SPECIAL SYMPOSIUM: MODERATOR: STEWART B. DUNSKER
Congenital Cranial Base & Spinal Problems

Panelists:

John C. VanGilder: Iowa City, IA John A. Jane: Charlottesville VA Charles J. Hodge, Jr.: Syracuse,NY

Albert L. Rhoton: Gainesville, FL

Harold J. Hoffman: Toronto, CA Paul H. Chapman: Boston, MA

1:00 p.m. ADJOURNMENT

Craniovertebral Anomalies
Craniofacial Anomalies
Syringomyella and the Chiari
Malformation; Mechanisms
Syringomyella and the Chiari
Malformation; Treatment
Tethered Cord
Spinal Lipomas

### NOTES

### RESIDENTS PAPER AWARD WINNERS

#### WINNER

#### MARY LOUISE HLAVIN, M.D.

# DEPARTMENT OF NEUROSURGERY CASE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE CLEVELAND, OHIO

MOLECULAR STRUCTURE AND FUNCTIONAL TESTING OF THE HUMAN L1 CELL ADHESION MOLECULE: AN INTERSPECIES COMPARISON

#### RUNNER UP

G. ALEXANDER WEST, M.D.

DEPARTMENT OF NEUROSURGERY HARBORVIEW MEDICAL CENTER SEATTLE, WASHINGTON

EFFECTS OF EXTERNAL PH ON IONIC CURRENTS IN SMOOTH MUSCLE CELLS FROM THE BASILAR ARTERY OF THE GUINEA PIG

#### **ACADEMY AWARD WINNERS**

Paul M. Lin	1955
Hubert L. Rosomoff	1956
Byron C. Pevehouse	1957
Norman Hili	1958
Jack Stern	1959
Robert Ojemann	1960
Lowell E. Ford	1962
Charles H. Tator	1963
Earle E. Crandall	1964
Stephen Mahaley, Jr.	1965
Chun Ching Kao	1966
John P. Kapp	1967
Yoshio Hosobuchi	1968
Gary G. Ferguson	1970
Richard I. Pressley	1971
David G. McLeone	1972
Arden F. Reynolds,Jr.	1973
Richard I. Rapport	1974
Andrew G. Shetter	1975
John F. Howe	1976
Howard W. Blume	1977
Howard J. Senter	1978
Elisabeth M. Post	1979
David Dubulsson	1980
Dennis A. Turner	1981
Marc R. Mayberg	1982
David S. Baskin	1983
Kevin J. Kiwak	1984
Terry Lichtor	1985
Michael G. Nosko	1986
Joseph R. Madsen	1987
James T. Rutka	1988
Christopher D. Heffner	1989
Scott I. Gingold	1990
Mary Louise Hiavin	1991
	1 1

**GUEST OF** GUEST **DANIEL L. BARROW GEORGE T. TINDALL** Atlanta GA KIM J. BURCHIEL THE ACADEMY Portland, OR **MAURICE COLLADA** DAVID G. KLINE Salem OR JAMES C. COLLIAS **NICHOLAS T. ZERVAS** Hartford CT **ROBERT C. DAUSER** JULIAN T. HOFF Ann Arbor MI TIMOTHY J. EBNER **ROBERTO HEROS** Minneapolis MN **EDUARDO EIDELBERG** JIM L. STORY San Antonio TX RUDOLPH FAHLBUSCH **NICHOLAS T. ZERVAS** Erlangan, Germany ROBERT C.A. FREDERICKSON JAMES T. ROBERTSON Beachwood OH JAMSHID GHAJAR RICHARD A.R. FRASER New York NY JOSEPH H. HAHN RALPH G. DACEY Cleveland OH WALTER A. HALL LYLE A. FRENCH

RUSSELL H. PATTERSON, JR

Minneapolis MN

**NICHOLAS HOPKINS** 

**Buffalo NY** 

**GUEST GUEST OF** MARK JONES CHARLES HODGE,JR. Syracuse NY PATRICK J. KELLY THORALF SUNDT Rochaster MN CHRISTOPHER LOFTUS JOHN VAN GILDER lows City IA JOHN C. MULLEN SHELLEY N. CHOU Minneapolis MN STEVEN E. MURK JIM L. STORY San Antonio TX **EDWARD NEUWELT CLARK WATTS** Portland OR THOMAS O. OESTERLING JAMES T. ROBERTSON Beachwood OH JON ROBERTSON JAMES T. ROBERTSON Memphis TN ROBERT H. ROSENWASSER WILLIAM A. BUCHHEIT Philadelphia PA WARREN R. SELMAN **ROBERT A. RATCHESON** Cleveland OH **EDWARD SELJESKOG ROBERTO HEROS** Minneapolis MN

CHARLES H. TATOR Toronto CA

**HAROLD HOFFMAN** 

ELLIS B. KEENER

### PAST PRESIDENTS

DEAN H. ECHOLS	1938-39
SPENCER BRADEN	1940
JOSEPH P. EVANS	1941
FRANCIS MURPHEY	1942
FRANK H. MAYFIELD	1943
A. EARL WALKER	1944
BARNES WOODHALL	1946
WILLIAM S. KEITH	1947
HOWARD A. BROWN	1948
JOHN RAAF	1949
E. HARRY BOTTERELL	1950
WALLACE B. HAMBY	1951
HENRY G. SCHWARTZ	1952
J.LAWRENCE POOL	1953
	1954
RUPERT B. RANEY	1955
DAVID L. REEVES STUART N. ROWE	1956
ARTHUR R. ELVIDGE	1957
	1958
JESS D. HERRMANN	1959
EDWIN B. BOLDREY	1960
GEORGE S. BAKER	1961-62
C.HUNTER SHELDEN	
SAMUEL R. SNODGRASS THEODORE B. RASMUSSEN	1964
EDMUND J. MORRISSEY	1965
	1966
GEORGE MALTBY GUY L. ODOM	1967
JAMES G. GALBRAITH	1968
ROBERT H. PUDENZ	1969-70
WILLIAM B. SCOVILLE	1971
ROBERT L. McLAURIN	1972
LYLE A. FRENCH	1973
BENJAMIN B. WHITCOMB	1974
BENJAMIN B. WHITCOMB	1975
JOHN R. GREEN	1976
WILLIAM H. FEINDEL	1977
WILLIAM H. SWEET	1978
ARTHUR A. WARD	1979
ROBERT B. KING	• • •
EBEN ALEXANDER, JR.	1981
EBEN ALEXANDER, JR. JOSEPH RANSOHOFF II BYRON C. PEVEHOUSE SIDNEY GOLDRING RUSSEL H. PATTERSON, JR.	1982
RIHON C. PEVENOUSE	1983
SIDNEY GOLDKING	1984
RUSSEL H. PATTERSON, JR.	1985
INUMAS LANGETTI	1986
PHANOR L. PEROT, JR.	1987
SHELLEY N. CHOU	1988
JAMES T. ROBERTSON	1989
THORALF M. SUNDT	1989
ROBERT G. OJEMANN	1880

#### PAST VICE-PRESIDENTS

FRANCIS MURPHEY	1941
WILLIAM S. KEITH	1942
IOUN DAAF	1042
DUDGOT D. DANEY	1043
HUPERI B. KANET	1944
ARTHUR R. ELVIDGE	1946
JOHN RAAF	1947
ARTHUR R. ELVIDGE	1948
F. KEITH BRADFORD	1949
DAVID L. REEVES	1950
HENRY G. SCHWARTZ	1951
J. LAWRENCE POOL	1952
RUPERT R. RANEY	1053
DAVID I REEVES	1055
STILART N. DOWE	1004
JECC D UEDDWANN	1955
JESS D. HEHHMANN	1956
GEORGE S. BAKER	1957
SAMUEL R. SNODGRASS	1958
C. HUNTER SHELDEN	1959
EDMUND MORRISEY	1960
DONALD F. COBURN	1961-62
EREN ALEXANDER, JR.	1963
GEORGE I MALTRY	1064
WILLIAM S. KEITH JOHN RAAF RUPERT B. RANEY ARTHUR R. ELVIDGE JOHN RAAF ARTHUR R. ELVIDGE F. KEITH BRADFORD DAVID L. REEVES HENRY G. SCHWARTZ J. LAWRENCE POOL RUPERT B. RANEY DAVID L. REEVES STUART N. ROWE JESS D. HERRMANN GEORGE S. BAKER SAMUEL R. SNODGRASS C. HUNTER SHELDEN EDMUND MORRISEY DONALD F. COBURN EBEN ALEXANDER, JR. GEORGE L. MALTBY ROBERT PUDENZ FRANCIS A. ECHLIN BENJAMIN WHITCOMB HOMER S. SWANSON AUGUSTUS MCCRAVEY	1066
EDANCIC A FOULIN	1903
PENIAMM WUTOOM	1966
BENJAMIN WHITCOMB	1967
HOMER S. SWANSON	1968
AUGUSTUS McCRAVEY	1969-70
EDWARD W. DAVIS	1971
HOMER S. SWANSON AUGUSTUS McCRAVEY EDWARD W. DAVIS JOHN R. GREEN GEORGE J. HAYES RICHARD L. DeSAUSSRE ERNEST W. MACK	1972
GEORGE J. HAYES	1973
RICHARD L. DeSAUSSRE	1974
ERNEST W. MACK	1075
ERNEST W. MACK FRANK E. NULSEN ROBERT S. KNIGHTON ROBERT G. FISHER H.T. BALLANTINE, JR. GEORGE EHNI	1076
POREDI & KNIGHTON	1070
BORERT O CIOUER	1977
RUBERT G. FISHER	1978
H.T. BALLANTINE, JR.	1979
GEORGE EHNI	1980
COURTLAND H. DAVIS, JR. JOHN F. MULLAN	1981
JOHN F. MULLAN	1982
HUGO RIZZOLI	1983
JAMES W. CORRELL	1984
E D MENDDIAN	400-
GRIFFITH R HADRI III	1005
Ellic D Veenen	1900
PODERT ORGANIA	1987
HUBERT GROSSMAN	1988
GRIFFITH R. HARSH III ELLIS B. KEENER ROBERT GROSSMAN JIM STORY JOHN A. JANE	1988
JOHN A. JANE	1990

### PAST SECRETARY-TREASURER

FRANCIS MURPHEY	1938-40
A.EARL WALKER	1941-43
C.ERICKSON	1944-47
WALLACE B. HAMBY	1948-50
THEODORE B. RASMUSSEN	1951-53
EBEN ALEXANDER, JR.	1954-57
ROBERT L. McLAURIN	1958-62
EDWARD W. DAVIS	1963-65
ROBERT G. FISHER	1966-68
BYRON C. PEVEHOUSE	1969-72

#### PAST SECRETARY

BYRON C. PEVEHOUSE	1973
RUSSEL H. PATTERSON, JR.	1974-76
PHANOR L. PEROT, JR.	1977-80
JOHN T. GARNER	1981-83
JAMES T. ROBERTSON	1984-86
NICHOLAS T. ZERVAS	1987-89
WILLIAM A. BUCHHEIT	1989-

### PAST TREASURER

RUSSEL H. PATTERSON, JR.	1973
PHANOR L. PEROT, JR.	1974-76
JOHN T. GARNER	1977-80
JAMES T. ROBERTSON	1981-83
NICHOLAS T. ZERVAS	1984-86
WILLIAM A. BUCHHEIT	1987-89
JULIAN T. HOFF	1990-

#### PAST MEETINGS OF THE ACADEMY

HOTEL NETHERLAND PLAZA, CINCINNATI, OHIO ROOSEVELT HOTEL, NEW ORLEANS, LOUISIANA TUDOR ARMS HOTEL, CLEVELAND, OHIO MARK HOPKINS HOTEL, SAN FRANCISCO AND AMBASSADOR HOTEL

LOS ANGELES, CALIFORNIA
THE PALMER HOUSE, CHICAGO, ILLINOIS
HART HOTEL,BATTLE CREEK, MICHIGAN
ASHFORD GENERAL HOSPITAL,WHITE SULPHUR

SPRINGS, WEST VIRGINIA THE HOMESTEAD, HOT SPRINGS, VIRGINIA BROADMOOR HOTEL, COLORADO SPRINGS,

COLORADO
WINDSOR HOTEL, MONTREAL, CANADA
BENSON HOTEL, PORTLAND, OREGON
MAYO CLINIC, ROCHESTER, MINNESOTA
SHAMROCK HOTEL, HOUSTON, TEXAS
WALDORF-ASTORIA HOTEL

NEW YORK CITY SEPTEMBER 29-OCTOBER 1,1952
BILTMORE HOTEL,SANTA BARBARA, CALIFORNIA OCTOBER 12-14,1953
BROADMOOR HOTEL, COLORADO SPRINGS,

COLORADO
THE HOMESTEAD, HOT SPRINGS, VIRGINIA
CAMELBACK INN, PHOENIX, ARIZONA
THE CLOISTER, SEA ISLAND, GEORGIA
THE ROYAL YORK HOTEL, TORONTO, CANADA
DEL MONTE LODGE, PEBBLE BEACH, CALIFORNIA
COPLEY SHERATON PLAZA.BOSTON.

MASSACHUSETTS
ROYAL ORLEANS, NEW ORLEANS, LOUISIANA
EL MIRADOR, PALM SPRINGS, CALIFORNIA
THE KEY BISCAYNE, MIAMI, FLORIDA
TERRACE HILTON HOTEL, CINCINNATI, OHIO
FAIRMONT HOTEL & TOWERS,
SAN FRANCISCO, CALIFORNIA
THE KEY BISCAYNE, MIAMI, FLORIDA
BROADMOOR HOTEL, COLORADO SPRINGS,
COLORADO

COLORADO
ST. REGIS HOTEL, NEW YORK CITY
CAMINO REAL HOTEL, MEXICO CITY
SAHARA-TAHOE HOTEL, STATELINE, NEVADA
NEW COLLEGE, OXFORD, ENGLAND

OCTOBER 28-29,1938 OCTOBER 27-29,1939 OCTOBER 21-22,1940

NOVEMBER 11-15,1941 OCTOBER 16-17,1942 SEPTEMBER 17-18,1943

SEPTEMBER 7-9, 1944 SEPTEMBER 9-11, 1946

OCTOBER 9-11,1947 SEPTEMBER 20-22,1948 OCTOBER 25-27,1949 SEPTEMBER 28-30,1950 OCTOBER 4-6,1951

OCTOBER 21-23,1954 OCTOBER 27-29,1955 NOVEMBER 8-10,1956 NOVEMBER 11-13,1957 NOVEMBER 6-8,1958 OCTOBER 18-21,1959

OCTOBER 5-8,1960 NOVEMBER 7-10,1962 OCTOBER 23-26,1963 NOVEMBER 11-14,1964 OCTOBER 14-16,1965

OCTOBER 17-19,1966 NOVEMBER 8-11,1967

OCTOBER 6-8,1968 SEPTEMBER 21, 1969 NOVEMBER 18-21,1970 SEPTEMBER 26-29,1971 SEPTEMBER 4-7,1972 HUNTINGTON-SHERATON HOTEL. NOVEMBER 14-17,1973 PASADENA, CALIFORNIA SOUTHAMPTON PRINCESS HOTEL. SOUTHAMPTON, BERMUDA **NOVEMBER 6-9.1974** THE WIGWAM(LITCHFIELD PARK). PHOENIX ARIZONA **NOVEMBER 5-8.1975** MILLS HYATT HOUSE. CHARLESTON, SOUTH CAROLINA NOVEMBER 10-13,1976 **NOVEMBER 2-5,1977** MAUNA KEA BEACH HOTEL, KAMUELA,HAWAII HOTEL BAYERISCHER HOF, MUNICH, GERMANY OCTOBER 2-25,1978 HYATT REGENCY, MEMPHIS, TENNESSEE NOVEMBER 76-10.1979 WALDORF ASTORIA, NEW YORK, NEW YORK OCTOBER 1-4,1980 SHERATON PLAZA, PALM SPRINGS, CALIFORNIA NOVEMBER 1-4,1981 RITZ-CARLTON HOTEL, BOSTON MASSACHUSETTS OCTOBER 10-13.1982 THE LODGE AT PEBBLE BEACH, CALIFORNIA OCTOBER 23-26,1983 THE HOMESTEAD, HOT SPRINGS, VIRGINIA OCTOBER 17-20.1984 THE LINCOLN HOTEL POST OAK, HOUSTON, TEXAS OCTOBER 27-30.1985 THE CLOISTER, SEA ISLAND, GEORGIA **NOVEMBER 5-8.1986 HYATT REGENCY, SAN ANTONIO, TEXAS** OCTOBER 7-10,1987 OMNI NETHERLAND PLAZA, CINCINNATI, OHIO SEPTEMBER 13-17.1988 LOEWS VENTANA CANYON RESORT. SEPTEMBER 27-OCTOBER 1, 1989 TUCSON, ARIZONA

# MEMBERSHIP LIST 1991 AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

OCTOBER 2-7,1990

HONORARY MEMBERS (5)	ELECTED
GUY LAZORTHES 26 Rue D Auriol 31 Toulouse, France	1973
VALENTINE LOGUE (Anne) 16 Rowan Road Hammersmith London W6 7DU England	1974

AMELIA ISLAND PLANTATION
AMELIA ISLAND,FL

GOSTA NORLEN (Gunvor) Linnegaten 35 IV 11447 Stockholm, Sweden	1973
BERNARD PERTUISET(Francoise) Hopital de la Pitie 83 Boulevard de l'Hopital 75651 Paris, Cedex 13 France	1986
KEIJI SANO (Yaeko) Teikyo University Hospitai 2-11-1 Kaga Itabashi-ku Tokyo 173, Japan	1975

SENIOR MEMBERS (60)	ELECTED
EBEN ALEXANDER, JR. (Betty) Bowman Gray School of Medicine 300 South Hawthorne Winston-Salem, North Carolina 27103	1950
GEORGE S. BAKER (Enid) 607 Litchfield Road Litchfield Park, Arizona 85340	1940
H. THOMAS BALLANTINE, JR. (Elizabeth) Massachusetts General Hospital 15 Parkman Street - ACC 312 Boston, Massachusetts 02114	1951
E. HARRY BOTTERELL (Margaret) 2 Lakeshore Boulevard Kingston, Ontario, Canada K7M 4J6	1938
HARVEY CHENAULT (Bille) 6340 Briar Hill Road Paris, Kentucky 40361	1949
SHELLEY N. CHOU (Jolene) University of Minnesota Medical Center 420 Delaware Street S. E., Box 96 Minneapolis, Minnesota 55455	1974
GALE G. CLARK 12621 Brookpark Road Oakland, California 94619	1970
W. KEMP CLARK (Fern) 5323 Harry Hines Boulevard Dallas, Texas 75235	1970
WILLIAM F. COLLINS, JR. (Gwendolyn) Yale University School of Medicine 333 Cedar Street New Haven, Connecticut 06510	1963

COURTLAND H. DAVIS, JR. (Carolyn) Bowman-Gray School of Medicine Winston-Salem, North Carolina 27103	1967
RICHARD DESAUSSURE, JR. (Phyllis) 920 Madison Avenue Memphis, Tennessee 38103	1962
DONALD F. DOHN (Carolyn) Cleveland Clinic Florida 3000 West Cypress Creek Road Ft. Lauderdale, Florida 33309	1968
R.M.PEARDON DONAGHY (Francis) P.O. BOX 5035, Road 1 Horn of the Moon Road Montpelier, Vermont 05602	1970
CHARLES G. DRAKE (Ruth) University Hospital London, Ontario, Canada N6A 5A5	1958
DEANE H. ECHOLS (Fran) 1550 Second Street New Orleans, Louisiana 70130	Founder
ROBERT FISHER (Connie) Box 846, Star Rt. 1 Bristol, New Hampshire 03222	1957
ELDON L. FOLTZ (Catherine) UCI Medical Center Division of Neurosurgery 101 City Drive, South Orange, California 92668	1960
LYLE A FRENCH (Gene) P.O. Box 1007 Pauma Valley, California 92061	1954

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WILLIAM F. MEACHAM (Alice) 539 Medical Arts Bidg. Nashville, Tennessee 37212	1952
FRANCIS MURPHEY (Marge) 3951 Gulf Shore Blvd. North Naples, Florida 33940	Founder
BLAINE S. NASHOLD, JR., (Irene) Duke University Medical Center Durham, North Carolina 27710	1967
GUY L. ODOM (Mataline) 2812 Chelsea Circle Durham, North Carolina 27707	1946
BYRON C. PEVEHOUSE (Lucy) 2351 Clay Street San Francisco CA 94115	1964
J. LAWRENCE POOL (Angeline) P.O. Box 40 West Cornwall, Connecticut 06796	1940
ROBERT W. PORTER (Aubrey Dean) 6461 Bixby Hill Road Long Beach, California 90815	1962
ROBERT H. PUDENZ (Rita) 574 Garfield Avenue South Pasadena, California 91030	1943
JOHN RAAF(Lorene) 1120 N.W. 20th Avenue, #100 Portland, Oregon 97209	Founder
AIDEN A. RANEY (Mary) 2010 Wilshire Boulevard Los Angeles, California 90057	1946
B. RAMAMURTHI (Indira) 2nd Main Road G.i.T. Colony Madras 4, India 600 004	1966

JOSEPH RANSOHOFF II (Lori) New York University Medical Center 550 First Avenue New York, New York 10016	1965
THEODORE B. RASMUSSEN (Catherine) 29 Surrey Drive Montreal, Quebec, Canada H3P 1B2	1947
HENRY G. SCHWARTZ (Reedle) Washington University School of Medicine 4901 Barnes Hospital Plaza St. Louis, Missouri 63110	1942
C. HUNTER SHELDEN (Elizabeth) Huntington Medical Research Institute 10 Pico Street Pasadena, California 91105	1941
ANTHONY SUSEN (Phyllis) 504 Remora Circle Fripp Island, South Carolina 29220	1965
WILLIAM H. SWEET (Elizabeth) 309 Goddard Avenue Brookline, Massachusetts 02146	1950
JOHN TYTUS (VirgInla) P.O. Box 279 Arlington, Washington 98223	1967
ALFRED UIHLEIN (Ione) P.O. Box 765 Vail, Colorado 81658	1949
A. EARL WALKER (Agnes) 1445 Wagontrain Drive, S.E. Albuquerque, New Mexico 87123	1938
ARTHUR A. WARD, JR. (Janet) 4001 N.E. Belvoir Place Seattle, Washington 98105	1953

EXUM WALKER (Nellie) 490 Peachtree Street, N.E. Atlanta, Georgia 30308	1938
W. KEASLEY WELCH (Elizabeth) 25 Gould Road Waban, MA 02168	1957
BENJAMIN B. WHITCOMB (Margaret) RFD Box 124 Surrey, Maine 04684	1947
LOWELL E. WHITE, JR. (Margie) University of South Alabama Division of Neuroscience Mobile. Alabama 36688	1971

ACTIVE MEMBERS (93)	ELECTED
MICHAEL J. APUZZO (Helene) 1200 N. State Street Los Angeles, California 90030	1988
JAMES I. AUSMAN (Carolyn) Henry Ford Hospital 2799 West Grand Boulevard Detroit, Michigan 48202	1978
DONALD P.BECKER (Marie) UCLA Medical Center Department of Neurosurgery Rm 74-140 Chs 405 Hilgard Los Angeses, California 90024	1990
GILLES BERTRAND (Louise) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 1B4	1967
PETER M. BLACK (Katherine) Brigham and Women's Hospital 75 Francis Street Boston MA 02115	1988
ROBERT S. BOURKE (Mariene) 5802 Nicholson Lane Rockville, Maryland 20852-2967	1983
JERALD S. BRODKEY (Arielle) 24755 Chagrin Boulevard Suite #205 Beachwood, Ohio 44122	1977
WILLIS E. BROWN, JR. (Ann) Division of Neurosurgery The University of Texas Health Science Ctr. 7703 Floyd Curl Drive San Antonio, Texas 78284-7843	1984

DEREK A. BRUCE (Frances) 7777 Forrest Lane #C703 Dallas, Texas 75230	1984
WILLIAM A. BUCHHEIT 3401 North Broad Street Philadelphia, Pennsylvania 19140	1980
WILLIAM F. CHANDLER (Sue) 2124D/338 Taubman Center 1500 East Medical Ctr. Drive Ann Arbor Mi 48109	1989
PAUL H. CHAPMAN (Tansy) Department of Neurosurgery Massachusetts General Hospital Boston, Massachusetts 02114	1983
EDWARD S. CONNOLLY (Elise) Ochsner Clinic 1514 Jefferson Highway New Orleans, Louisiana 70121	1973
JAMES W. CORRELL (Cynthia) 710 West 168th Street New York, New York 10032	1966
ROBERT .CROWELL(Mary) Massachusetts General Hospital Chief, Cerebrovascular Section Department of Neurosurgery/ACC 312 Boston, Massachusetts 02114	1990
RALPH G. DACEY(Corinne) Washington School of Medicine Division of Neurosurgery Barnes Hospital Plaza St. Louis, Missouri 63110	1990

ARTHUR L. DAY (Dana) University of Florida Health Ctr Department of Neurosurgery Box J 265 Gainesville, Florida 32610	1990
STEWART B. DUNSKER (Ellen) Mayfield Neurological Institute 506 Oak Street Cincinnati, Ohio 45219	1975
HOWARD M. EISENBERG (Janet) The University of Texas Medical Branch Division of Neurosurgery Galveston, Texas 77550	1985
WILLIAM H. FEINDEL (Faith) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 2B4	1959
EUGENE FLAMM (Susan) Hospital of the University of Pennsylvania 3400 Spruce Street Philadelphia,Pennsylvania 19104	1979
RICHARD A.R. FRASER (Sarah Anne) 525 East 68th Street New York, New York 10021	1976
JOHN T. GARNER (Candace) 50 Allesandro Piace Suite 400 Pasadena, California 91105	1971
HENRY GARRETSON (Marianna) Health Sciences Center 316 MDR Bldg. University of Louisville Louisville, Kentucky 40292	1973

ROBERT G. GROSSMAN (Ellin) Baylor College of Medicine One Baylor Place Houston, Texas 77030	1984
ROBERT GRUBB (Julia) Washington University School of Medicine 4901 Barnes Hospital Plaza St. Louis, Missouri 63110	1985
GRIFFITH R. HARSH, III (Craig) Division of Neurosurgery UBA Station Birmingham, Alabama 35294	1980
MARK PETER HEILBRUN (Robyn) Division of Neurosurgery #3B320 University of Utah Medical Center 50 North Medical Drive Salt Lake City, Utah 84132	1984
E. BRUCE HENDRICK (Gloria) Hospital for Sick Children 555 University Avenue, Room 1502 Toronto, Ontario, Canada M5G 1X8	1968
ROBERTO C. HEROS (Deborah) University of Minnesota Medical Center 420 Southwest Delaware Street Box 96 Minneapolis, MN 55455	1985
CHARLES HODGE (Linda) 750 East Adams Street Syracuse, New York 13210	1982
JULIAN HOFF (Diane) Department of Neurosurgery University of Michigan Ann Arbor, Michigan 48109	1975

HAROLD HOFFMAN (Jo Ann) The Hospital for Sick Children Sulte 1502, 555 University Avenue Toronto, Ontario, Canada M5G 1X8	1982
EDGAR M. HOUSEPIAN (Marion) The Neurological Institute 710 West 168th Street New York, New York 10032	1976
ALAN R. HUDSON (Susan) St. Michael's Hospital 38 Shuter Street Toronto, Ontario, Canada M5B 1A6	1978
JOHN A. JANE (Noella) Department of Neurosurgery, Box 212 University of Virginia Charlottesville, Virginia 22908	1982
ELLIS B. KEENER (Ann) 915 East Lake Drive, N.W. Gainesville, Georgia 30506	1978
DAVID KELLY, JR. (Sally) Bowman Gray School of Medicine 300 S. Hawthorne Winston-Salem, North Carolina 27103	1975
GLENN W. KINDT (Charlotte) Division of Neurosurgery Box C-307 University of Colorado Medical Center 4200 East 9th Avenue Denver, Colorado 80262	1977
WOLFF M. KIRSCH (Marie-Claire) Chief of Neurosurgery Univ. of New Mexico Medical School Albuquerque, New Mexico 87131	1971

DAVID G. KLINE Louisiana State University Medical Center 1542 Tulane Avenue New Orleans, Louisiana 70112	1972
RICHARD S. KRAMER (Mollie) Duke Hospital Medical Center Durham, North Carolina 27710	1978
THEODORE KURZE University of Pittsburgh Department of Neurosurgery 9402 Presbyterian University Hospital 230 Lothrop Street Pittsburgh, PA 15213	1967
SANFORD LARSON (Jackie) Medical College of Wisconsin 8700 W. Wisconsin/Neurosurgery Milwaukee Wi 53226	1989
EDWARD R. LAWS, JR. (Peggy) George Washington Medical Center 2150 Pennsylvania Ave. NW Washington, D. C. 20037	1983
DONLIN M. LONG (Harriet) Department of Neurological Surgery Johns Hopkins Medical School 601 N. Wolfe Baltimore, Maryland 21205	1983
ALFRED J. LUESSENHOP (Frances) Georgetown University Hospital 3800 Reservoir Road Washington, D. C. 20007	1976
JOE MAURICE McWHORTER (Barbara) Bowman Gray School of Medicine Winston-Salem NC 27103	1989

LEONARD MALIS (Ruth) 1176 Fifth Avenue New York, New York 10029	1973
ROBERT L. MARTUZA (Jill) Massachusetts General Hospital Fruit Street Boston MA 02114	1989
RICHARDB.MORAWETZ (MaryJean) Division of Neurosurgery University Station Birmingham, Alabama 35294	1990
JOHN F. MULLAN (Vivian) 5844 Stoney Isle Avenue Chicago, Illinois 60637	1963
FRANK E. NULSEN (Ginny) 32 10th Avenue, South Naples, Florida 33940	1956
GEORGE OJEMANN (Linda) 6424 E. Mercer Way Mercer island, Washington 98040	1975
ROBERT G. OJEMANN (Jean) Neurosurgery Service Massachusetts General Hospital Boston, Massachusetts 02114	1968
ANDRE OLIVIER (Nichole) 3801 University Street Suite #107 Montreal PQ H3A 2B4	1989
BURTON ONOFRIO (Judith) Mayo Clinic Rochester, Minnesota 55905	1975

RUSSEL H. PATTERSON, JR. (Julie) New York Hospital 525 East 68th Street New York, New York 10021	1971
SYDNEY J. PEERLESS (Ann 1501 N. W. 9th Avenue Miami, Fiorida 33136	1977
PHANOR L. PEROT, JR. Department of Neurosurgery Medical University of South Carolina 171 Ashley Avenue Charleston, South Carolina 29425	1970
DAVID G. PIEPGRAS (Jane) Mayo Clinic 200 First Street, S.W. Rochester, Minnesota 55905	1987
DONALD O. QUEST (Ilona) The Neurological Institute 710 West 168th Street New York, New York 10032	1968
ROBERT A. RATCHESON (Peggy) University Hospital 2074 Abington Road Cleveland, Ohio 44106	1986
ALBERT L. RHOTON, JR. (Joyce) University of Florida, Box J265 Department of Neurosurgery Gainesville, Florida 32610	1984

J. CHARLES RICH, JR. (Jasmine) 324 10th Avenue #206 Salt Lake City, Utah 84103	1987
HUGO RIZZOLI (Helen) 2150 Pennsylvania Avenue, N.W. Washington, D.C. 20037	1973
THEODORE S. ROBERTS (Joan) Department of Neurological Surgery University Hospital 1959 Pacific Avenue, N.E., RI 20 Seattle, Washington 98195	1976
JAMES T. ROBERTSON (Valeria) Department of Neurosurgery University of Tennessee, Memphis 956 Court Avenue Memphis, Tennessee 38163	1971
WILLIAM SHUCART (Laura) New England Medical Ctr. #178 750 Washington Street Boston MA 04401	1989
FREDERICK A. SIMEONE (Kate) Pennsylvania Hospital 800 Spruce Street Philadelphia, Pennsylvania 19107	1981
JAMES C. SIMMONS (Vanita) 920 Madison Avenue, 201-N Memphis, Tennessee 38103	1975
KENNETH R. SMITH, JR. (Marjorle) St. Louis University Hospital 3635 Vista Avenue St. Louis, Missouri 63110-2500	1987

ROBERT R. SMITH (Helen) University of Mississippi Medical Ctr. Department of Neurosurgery Jackson MS 39216	1989
DENNIS SPENCER (Susan) 333 Cedar Street New Haven CT 06510	1989
BENNETT M. STEIN (Bonita) 710 West 168th street New York, New York 10032	1970
JIM L. STORY (Joanne) Division of Neurosurgery The University of Texas Health Science Ctr 7703 Floyd Curl Drive San Antonio, Texas 78284-7843	1972
THORALF M. SUNDT, JR. (Lois) Department of Neurosurgery Mayo Clinic Rochester, Minnesota 55905	1971
RONALD R. TASKER (Mary) Toronto General Hospital Room 215, 14th Floor 200 Elizabeth Street Toronto, Ontario, Canada M5G 2C4	1971
JOHN TEW, JR. (Susan) 506 Oak Street Cincinnati, Ohio 45219	1973
GEORGE TINDALL (Suzie) Emory University School of Medicine Division of Neurosurgery 1365 Clifton Road, N.E. Atlanta, Georgia 30322	1968

SUZIE C. TINDALL (George) Emory University School of Medicine Division of Neurosurgery 1327 Clifton Road, N.E. Atlanta, Georgia 30322	1990
JOHN C. VAN GILDER (Kerstin) University of Iowa Hospital Iowa City, Iowa 55242	1980
CLARK WATTS (Patty) One Hopital Drive Ste. N.522 Columbia, Missouri 65212	1975
BRYCE K. A. WEIR (Mary Lou) 2D2-24 Mackenzle Health Sciences Center 8440-112 Street Edmonton, Alberta, Canada T6G 2B7	1984
MARTIN H. WEISS (Debby) USC Medical Center 1200 North State Street Los Angeles, California 90033	1981
ROBERT WILKINS (Gloria)  Duke University Medical Center  Box 3807  Durham, North Carolina 27710	1973
CHARLES B. WILSON Department of Neurological Surgery University of California Medical Center Third and Parnassus San Francisco, California 94143	1966

ALLEN WYLER (Lily) University of Tennessee, Memphis Department of Neurosurgery Suite 201 920 Madison Avenue Memphis, Tennessee 38103	1990
DAVID YASHON (Myrna) St. Anthony Medical Center 1492 East Broad street Suite 1100 Columbus, Ohio 43205	1972
ALFRED BYRON YOUNG (Judy) University of Kentucky Medical Ctr. 800 Rose Street Division of Neurosurgery Lexington KY 40506	1989
RONALD F. YOUNG (Shella) University of California at Irvine 101 The City Drive South Orange, California 92668	1986
NICHOLAS T. ZERVAS (Thalia) Fruit Street Massachusetts General Hospital Boston, Massachusetts 02114	1972

### INACTIVE MEMBERS (2)

1972

M. STEPHEN MAHALEY, JR. (Jean) P.O. Box 1063 Maggie Valley, North Carolina 28751

JOHN P. KAPP (Lureese) 406 North Main Street Galax, Virginia 24333

# SENIOR CORRESPONDING MEMBERS (10)

JEAN BRIHAYE (Martine Van Geertruyden) 98 Ave. Des Franciscainn 1150 Bruxelles, Belgium	1975
KARL AUGUST BUSHE (Eva) Neurochirurgischen Klinik Josef-Schnelder-Strasse II D-8700 Wurzburg, West Germany	1971
JOHN HANKINSON (Nicki) Westacres Woolsington Hali Newcastle-Upon-Tyne England	1973
SHOZO ISHII Department of Neurosurgery Juntendo Medical College Tokyo 113, Japan	1975
HANS-PETER JENSEN (RETA) Neurochirurgische Universitatsklinik Kiel Welmarer Strasse 8 D-2300 Kiel/West Germany	1980
KATSUTOSHI KITAMURA (Yoshiko) Shinkokura Hospital 1-2-1 Kanada Kokurakita-Ku Kitakyushu, 803 Japan	1970
KRISTIAN KRISTIANSEN (Brit) Ulleval Hospital 0407 Osio, 4 Norway	1962
WILLIAM LUYENDIJK (Tony) Pr Bernhardiaan 60 Oegstgeest, The Netherlands	1973

B. RAMAMURTHI (Indira) 2nd Main Road G.I.T. Colony Madras 4, India 600 004

1966

KURT SHURMANN
Director
Neurochirurg
Univ-Klinik Mainz
Langenbeskstr 1
6500 Mainz, West Germany

1978

# CORRESPONDING MEMBERS (27)

LEIGH R. ATKINSON (Alexandra) 201 Wickham Terrace 4000 Brisbane, Qid. Australia	1989
FERNANDO CABIESES Inst. Peruano De Formento Educativo Av. Arenales 371, of. 501 Apartado 5254 Lima, Peru	1966
JUAN CARDENAS Insurgentes Sur 594 Av. Insurgentes Mexico City, Mexico 40	1966
LUC CALLIAUW (Dora) Bisschopdreef 53 8310 Brugge, Belgium	1988
JUAN C. CHRISTENSEN Ayacucho 2151 4 P Buenos Aires, Argentina	1970
GUISEPPE DALLE ORE (Glusi) Clinica Neurochirurgica Universita di Verona Piazzale Stefani 37100 Verona, Italy	1970
NOEL G. DAN Suite 5 Specialist Medical Center 235-285 New South Head Road Edgecliff 2027 Sydney, N.S.W. Australia	1989

JACQUES DEVILLIERS (Jeanne Marie) Department of Neurosurgery Groote Schuur Hospital Observatory 7925 Cape Town Republic of South Africa	1986
HANS ERICH DIEMATH (Karin) Landesnergenklinik Ignaz Harrer-Strasse 79 A-5020 Salzburg, Austria	1970
HERMANN DIETZ Neurosurgical Clinic Hannover School of Medicine Hannover 3000-61 West Germany	1980
VINKO DOLENC (Petra) Klinicki Bolnicki Ctr. Klinika Neurokirurgijo Zaleski C7 6100 Ljubljana, Yugoslavia	1988
JOHN F. GILLINGHAM (Judy) Royal Infirmary Lauriston Place Edinburgh, Scotland EH43 PB United Kingdom	1962
JAMIE G. GOMEZ (Lucy) V.I. Medical Foundation Bidg. #103 Charlotte Amnalie, St. Thomas U.S. Virgin Islands 00802	1975
SALVADOR GONZALEZ-CORNEJO (Rosalie) Av. Chapultepec Sur 130-204 Guadalajara, Mexico 44100	1982
ERNEST H. GROTE (Julie) Neurosurgery Department University Clinic, Calwer Strasse 7 7400 Tubingen, Federal Republic of Germany	1984

HAJIME HANDA (Hiroko) Hamamatsu Rosai Hospital 25 Shogen-Cho, Hamamatsu 430 Japan	1985
FABIAN ISAMAT (Marivi) Clinica Sagrade Familia Torras y Pujalt, 1 08022 Barcelona, Spain	1986
RICHARD JOHNSON Department of Neurological Surgery Royal Infirmary Manchester, England	1974
LAURI LAITINEN (Kerstin) Rosnedalsslingan 21 18633 Vallentuna Sweden	1971
FRANK MARGUTH Director, Department of Neurochlrurgischen Universitat Munchen Marchioninistrasse 15 8000 Munchen 70, West Germany	1978
PAUL MARINO, JR. (Milu) Rua Maestro Cardim, 808/814 S. Paulo-SP Brazil	1977
J. DOUGLAS MILLER Western General Hospital Crewe Rd. Edinburgh EH4 2XU Scotland	1988
KENICHIRO SUGITA Nagoya University School of Medicine 65 Tsumai-Cho, Showa-Ku Nagoya 466, Japan	1988

CHARAS SUWANWELA Chulalongkorn Hospital Medical School Bangkok, Thailand	1972
LINDSAY SYMON (Pauline) The National Hospital Queen Square London, WC1N 3BG England	1982
KINTOMO TAKAKURA University of Tokyo Hospital 7-3-1 Hongo, Bunkyu-ku Tokyo 113, Japan	1988
KJELD VAERNET (Ann) Department of Neurosurgery Rigshospitalet 9 Blegdamsvej 2100 Copenhagen, Denmark	1970
SIDNEY WATKINS The London Hospital Whitechapel, London E 1 England	1975
GAZI YASARGIL (Dianne) Neurosurgical Clinic University Hospital Ramistrasse 10 CH-8091 Zurich, Switzerland	1975

DECEASED MEMBERS		ELECTED
SIXTO OBRADOR ALCALDE Madrid, Spain (Honorary)	4/1978	1973
JAMES R. ATKINSON Phoenix, Arizona (Active)	2/1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	8/1973	1963
EDWIN B. BOLDREY San Francisco, California (Senior)	6/1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	7/1969	Founder
HOWARD A. BROWN Wainut Creek, California (Senior)	2/1990	1939
DONALD COBURN Wilmington, Delaware (Senior)	9/1988	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/1960	1942
EDWARD DAVIS Troutdale, Oregon (Senior)	10/1988	1949

FRANCIS ECHLIN New Paltz, New York (Senior)	4/1988	1944
GEORGE EHNI Houston, Texas (Senior)	9/1986	1964
ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1/1985	1939
THEODORE C. ERICKSON Madison, Wisconsin (Senior)	10/1986	1940
JOSEPH P. EVANS Kensington, Maryland (Senior)	5/1985	Founder
JOHN D. FRENCH Los Angeles, California (Senior)	1/1989	1951
JOHN R. GREEN Phoenix, Arizona (Senior)	1/1990	1953
WESLEY A. GUFTAFSON Jensen Beach, Florida (Senior)	7/1975	1942
HANNIBAL HAMLIN Providence, Rhode Island (Senior)	6/1982	1941
HENRY L. HEYL Hanover, New Hampshire (Senior)	3/1975	1951

OLAN HYNDMAN Iowa, City, Iowa (Senior)	6/1966	1942
KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	7/1976	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	3/1961	1951
WILLIAM KEITH Toronto, Canada (Senior)	12/1987 Fo	under
HUGO KRAYENBUHL Zurich, Switzerland (Honora <u>r</u> y)	1985	1974
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1/1980	1973
HERBERT LOURIE Syracuse, New York (Senior)	3/1987	1965
GEORGE L. MALTBY Boston, Massachusetts (Active)	1988	1942
DONALD D. MATSON Boston, Massachusetts (Active)	5/1969	1950
FRANK H. MAYFIELD Cincinnati, Ohio (Senior)	1/2/91 Fou	ınder
AUGUSTUS McCRAVEY Chattanooga, Tennessee (Senior)		1944

KENNETH G. McKENZIE Toronto, Ontario, Canada (Honorary)	2/1964	1960
JAMES M. MEREDITH Richmond, Virginia (Active)	12/1962	1946
W. JASON MIXTER Woods Hole, Massachusetts (Honorary)	3/1968	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	2/1986	1941
HANS-WERNER PIA Glessen, West Germany (Corresponding)	7/1986	1978
WILDER PENFIELD Montreal, Canada (Honorary)	4/1976	1960
HELMUT PENZHOLZ West Germany (Corresponding)	1985	1978
RUPERT B. RANEY Los Angeles, California (Active)	11/1959	1939
DAVID L. REEVES Santa Barbara, California (Senior)	8/1970	1939
DAVID REYNOLDS Tampa, Florida (Active)	4/1978	1964

R.C.L. ROBERTSON Houston, Texas (Senior)	2/1985	1946
STEWART N. ROWE Pittsburgh, Pennsylvania (Senior)	10/1984	1938
RICHARD C. SCHNEIDER Ann Arbor, Michigan (Senior)	6/1986	1970
WILLIAM B. SCOVILLE Hartford, Connecticut (Senior)	2/1984	1944
R. EUSTACE SEMMES Memphis, Tennessee (Honorary)	3/1982	1955
SAMUEL R. SNODGRASS Galveston, Texas (Senior)	8/1975	1939
GLEN SPURLING LaJolia, California (Honorary)	2/1968	1942
C. WILLIAM STEWART Montreal, Quebec, Canada (Corresponding)	1948	1948
HENDRIK SVIEN Rochester, Minnesota (Active)	6/1972	1957
HOMER S. SWANSON Atlanta, Georgia (Senior)	6/1987	1949

THOMAS A. WEAVER, JR. Dayton, Ohlo	19'85	1943
(Senior)		
BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941
FRANK WRENN Greenville, South Carolina (Active)	2/1990	1973

### **NOTES**

## NOTES